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      (FILE 'HOME' ENTERED AT 08:56:09 ON 21 JUL 2002)

      FILE 'MEDLINE, SCISEARCH, BIOSIS, CAPLUS' ENTERED AT 08:56:29 ON 21 JUL
      2002
L1         35003 S (INFLAMMATORY BOWEL DISEASE)
L2           326 S (MACROPHASE ELASTASE) OR MMP-12
L3             0 S L1(5A)L2
L4             1 S L1 AND L2

      FILE 'STNGUIDE' ENTERED AT 08:58:40 ON 21 JUL 2002

      FILE 'MEDLINE, SCISEARCH, BIOSIS, CAPLUS' ENTERED AT 09:05:54 ON 21 JUL
      2002
L5        373065 S L1 OR INFLAMMATION
L6             3 S L5(5A)L2
L7        645042 S L5 OR ULCER?
L8             5 S L7(5A)L2

      FILE 'STNGUIDE' ENTERED AT 09:09:00 ON 21 JUL 2002

      FILE 'MEDLINE, SCISEARCH, BIOSIS, CAPLUS' ENTERED AT 09:11:14 ON 21 JUL
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                E CHAKRAVARTI SHKTI/AU
L9            41 S E7
L10            0 S L9 AND L2
L11          575 S MACROPHAGE ELASTASE OR MMP-12
L12            0 S L9 AND L11
L13          575 S (MACROPHAGE ELASTASE) OR MMP-12
L14            0 S L13 AND L9
L15            4 S L9 AND L1

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Distinct expression profiles of stromelysin-2 (MMP-10), collagenase-3 (MMP-13), macrophage metalloelastase (MMP-12), and tissue inhibitor of metalloproteinases-3 (TIMP-3) in intestinal ulcerations

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Programmed expression of matrix metalloproteinases is involved in wound healing in various organs. We have previously demonstrated enhanced expression of collagenase-1, stromelysin-1, matrilysin, and tissue inhibitor of metalloproteinases (TIMP-1) in gastrointestinal ulcerations. To further define the role of matrix-degrading enzymes and their inhibitors in intestinal inflammation and ulcerations, the expression of stromelysin-2 (MMP-10), collagenase-3 (MMP-13), macrophage metalloelastase (HME, MMP-12), and TIMP-3 mRNAs was studied using in situ hybridization and immunohistochemistry in 38 samples representing ulcerative colitis, Crohn's disease, ischemic colitis, and normal intestine. As controls for normally healing intestinal wounds, 12 postoperative samples of rat experimental jejunal anastomoses were also examined. The colitis types studied did not essentially differ in their MMP expression. We found stromelysin-2 mRNA in laminin-5-positive and Ki-67-negative enterocytes bordering the ulcerations. HME was abundantly expressed by macrophages in the vicinity of shedding mucosal epithelium and beneath the necrotic surface of the ulcers. Collagenase- 3 and TIMP-3 were expressed by fibroblast-like cells deeper in the remodeling intestinal wall. Expression for stromelysin-2 and collagenase-3 was observed in granulation tissue, but not the epithelium, of the rat anastomoses. Our results suggest a role for stromelysin-2 in epithelial migration and for metalloelastase in macrophage movement and epithelial cell shedding.

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